Witness Name: Dr Charles Hay
Statement No: WITN3289026
Exhibits: None
Dated: 1 June 2020

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF DR CHARLES HAY

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated January 2020 in relation to criticisms from Mr GRO-B

I, Dr Charles Hay, will say as follows: -

Section 1: Introduction

1. Professor Charles Richard Morris Hay MBChB MD FRCP FRCPath

Consultant Haematologist Manchester Royal Infirmary since December 1994.
Director Manchester Adults Haemophilia Comprehensive Care Centre since December 1994
Senior Lecturer in Haematology Liverpool University and Director Liverpool Haemophilia Centre, Royal Liverpool Hospital 1987-1994.
Professor of Haemostasis and Thrombosis.
Director UK National Haemophilia Database since 2002.
Member UK Haemophilia Centre Doctors Organisation (UKHCDO) Regional Committee from 1987 and then Advisory Committee since 2007 (when the committee name changed).
Vice Chairman UKHCDO 1997 to 2005.
Chairman UKHCDO 2005-11.
I have produced a copy of my curriculum vitae and publications as an exhibit to previous statements to the Inquiry.

Background:

1. The Manchester Haemophilia Comprehensive Care Centre (Adults) is based in Manchester Royal Infirmary. This was the third largest Haemophilia Centre in the United Kingdom. It is now the second largest with >2500 registered patients with bleeding disorders. When I arrived in 1994, I was the only consultant specialising in adult Thrombosis and Haemostasis in the North West Region, assisted by a part-time clinical assistant. We now have four consultants with this specialism. In 1994, we had three Haemophilia Nurses, one of whom also did counselling and went into the community. There were no clinical research staff. There were no joint clinics and no formal liaison with any other supporting specialism (such as hepatology) or profession allied to medicine, such as physiotherapy when I arrived in December 1994. All the follow-up clinics were conducted in the Haemophilia Centre without the assistance of any junior staff. There was no internal training rotation for junior staff who covered other areas of haematology but not haemophilia. I was on call 1:1 i.e. 365 days a year except when away or on holiday.

2. In 1995 I introduced an internal training rotation for junior staff so that we had a registrar attached to Thrombosis and Haemostasis most of the time. I introduced weekly multidisciplinary meetings and arranged for Physiotherapy input for our patients. I rapidly established joint clinics for Orthopaedics and subsequently joint HIV clinics and joint obstetric clinics and later joint adolescent clinics with the paediatric service. There was liaison with Hepatology on a patient by patient basis throughout this period but not formalised in a joint-clinic. As we acquired more consultants specialising in Thrombosis and Haemostasis, first in 1999 and then in 2003 and in 2018, the patients were reallocated among the consultants. Almost all the HIV positive patients have remained with me and are jointly managed with Dr Ashish Sukthanker, Consultant HIV Physician. Patients with hepatitis C were managed by all the Thrombosis and Haemostasis Consultants.

3. I was Mr GRO-B Consultant Haematologist between 1995 and 1997, when he left the area. I was single-handed throughout that time. I have no access to Mr GRO-B

notes, which I have been informed were long since destroyed and unfortunately I have no recollection of him or the consultations to which he refers.

4. Mr GRO-B was attending the Centre at what was the busiest period for me, when I was managing a group of about 60 HIV positive patients at the early period of the HIV crisis. This was the year before triple therapy for HIV became established, and at a time when 15% of the UK HIV infected patients died in a single 12 month period, either from AIDS or advanced liver disease. It was a terrible time for the patients, their relatives, and the treating physicians. On average, at that time we had four inpatients being treated for AIDS complications. Thankfully, this came to an abrupt end when we introduced triple therapy in 1995. I have patients alive and healthy now who almost died at that time but who were successfully treated by the timely introduction of HIV triple therapy.

5. Hepatitis C therapy was in its infancy in 1995. Initially therapy was with Interferon three times a week for six months. Thus it is likely that this was the treatment offered in 1995. The response and relapse-rate with this treatment was very suboptimal. In later years Ribivarin was added in and then Interferon was changed to weekly Peginterferon. The response depended on Genotype. The response to Peginterferon and Ribivarin was very good for Genotypes 2 and 3, but only 40% overall for genotypes 1a and 1b. These genotypes are found all over the world. For example, Genotype 4 is found in sub-Saharan Africa. Genotyping was not available in 1995 and so I assume that our attempt to eradicate HCV was unsuccessful and that Mr GRO-B was probably genotyped and given further treatment in other centres.

6. Mr GRO-B says that he requested testing when he registered with our Centre in 1995. We would have tested him for HCV as a routine and offered treatment for his HCV if appropriate. From his description, he had already made efforts on his own behalf to obtain information before he consulted us.

Section 2: Responses to criticism of Mr GRO-B

7. Mr GRO-B says he was provided only with perfunctory and inadequate information about hepatitis C and its treatment.

8. I am sorry Mr GRO-B felt that the information provided was perfunctory and inadequate as this would not have been the intention or the ethic of the centre. My consultations were not characteristically short or perfunctory, which may be why my clinics generally last an hour or more, longer than those of my colleagues'. Treatment for HCV was in its infancy at that time. There was also limited knowledge about the efficacy and side-effect profile of the treatment. We would have warned Mr GRO-B about flu-like symptoms, and depression. The symptoms and side effects of subsequent combinations of Interferon with Ribivarin were significantly more severe than Interferon alone, with weight loss, anaemia, depression fatigue and general malaise occurring in most patients. The side effects varied significantly between patients but affected almost all patients to some degree. Unfortunately in order to be curative, it was treatment that had to be endured. These side effects would be described in detail to all patients considering treatment.

9. Genotyping of HCV was unavailable in 1995 and this limited what one could explain to patients. When the test became available, all HCV patients were genotyped. The prognosis, the response to treatment, and the length of treatment offered, were all informed by the HCV genotype and this was always explained to patients at that time. From his description, it appears he did not have the commonest genotype (1a or 1b) but one of the less aggressive and more responsive genotypes, probably genotype 2 or 3.

10. Although Mr GRO-B remembers the information as perfunctory, he appears to have absorbed the salient points about the potential long term outlook for untreated HCV. As time went by and our staffing level improved, we also provided the patients with more psychological support during their treatment for Hepatitis C, especially once we started to combine it with Ribivarin, which seemed to make the side effects more severe. Many patients suffered significantly with treatment side-effects and needed a lot of support. We also used to counsel patients and their partners before starting treatment because the direct treatment side-effects such as depression, which was often severe, chronic fatigue, and short-temper, would often put their relationships under strain.

Statement of Truth

I believe that the facts stated in this witness statement are true.

Signed GRO-C

Dated: 1 June 2020